POSTER

Characterization of the expression of pathogenic *Banana streak virus* sequences integrated in the genome of banana during genetic crosses.

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Banana streak virus (BSV) sequences are integrated in the nuclear genome of *Musa* species. Some of these BSV endogenous pararetrovirus (BSV EPRVs), restricted to *Musa balbisiana* chromosome (noted B), can give rise to infectious episomal BSV particles upon stress conditions such as inter-specific genetic crosses.
A genetic analysis of BSV incidence in a F1 triploid (*Musa* AAB) population produced by inter-specific hybridisation between virus-free diploid *M. balbisiana* (BB) and tetraploid *M. acuminata* (AAAA) parents showed that half of the F1 progeny expressed BSV particles. A PCR-based analysis determined that BSV EPRVs were restricted to the *M. balbisiana* genome only. A bulk AFLP segregant analysis revealed seven AFLP markers co-segregating with the presence of BSV infection and three with the absence of BSV infection. All of them were restricted to the *M. balbisiana* genome. Analysis of the segregation of these markers using a test-cross configuration allowed the construction of a genetic map of the linkage for the female parent (BB), containing the locus BEL associated with BSV infection in the F1 hybrid population. A monogenic allelic system conferring the role of carrier to the *M. balbisiana* parent seems to be involved in BSV appearance in this progeny.
Little is currently known about the real mechanisms underlying the genetic expression of pathogenic BSV EPRVs and their regulations. The role of methylation was investigated. Differential cytosine methylation patterns were searched in healthy and diseased F1-triploid (AAB) hybrids using the SD-AFLP/MSAP technique. The role of chromosomal rearrangements was also investigated, through a PCR-based analysis of both genomic DNA extracted from the progeny and BSV positive BAC clones of the *M. balbisiana* parent.
Among the thirteen DNA fragments obtained by SD-AFLP/MSAP, one corresponds to one AFLP marker closely located to the BEL locus. Differential PCR patterns were observed depending on the strain specific primers used, covering distinct parts of the genome of BSV, raising evidence for chromosomal rearrangements in diseased hybrids. This is corroborated by BAC clones of the PKW parent analysis.